

Cold tolerance and temperature regulation in birds

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RIASSUNTO

Resistenza al freddo e regolazione della temperatura corporea negli uccelli.

Gli uccelli, come i mammiferi, sono animali a temperatura corporea costante. Essi sono in grado di mantenere la propria temperatura normale di 40-42 °C con molti e vari mezzi di regolazione in quasi tutte le condizioni ambientali. Il controllo della temperatura avviene sia con mezzi esterni (ad esempio riparando in posti assolati o trovando rifugio nella neve) sia con mezzi propri, endotermici, aumentando cioè la produzione di calore. Il manto piumoso degli uccelli è un ottimo ostacolo contro la dispersione del calore, ma allorché esso diventa insufficiente, l'uccello deve al freddo aumentare la propria produzione di calore in primo luogo per mezzo del tremore muscolare. Gli uccelli non dispongono del tessuto adiposo bruno, produttore di calore tipico dei più piccoli mammiferi. Nonostante ciò sembra che almeno i galliformi producano ulteriore calore anche con mezzi non dipendenti dal tremore muscolare (= NST cioè "nonshivering thermogenesis"). Ciò non è comunque stimolato dal sistema nervoso simpatico, come accade nei mammiferi; dipende più probabilmente dal glucagone, uno degli ormoni pancreatici, nonché dall'ormone dell'accrescimento ipofisario. Allorché la temperatura esterna raggiunge valori al di sotto dello zero, insieme ai galliformi, anche molti piccoli passeracei possono rifugiarsi nella neve. In condizioni atmosferiche polari, gli uccelli più piccoli sembrano vincere la lunga e fredda notte cadendo nell'ipotermia: la temperatura corporea scende in maniera regolata di 10-15 °C al di sotto dei suoi valori normali.

Birds are found in a wide variety of habits and from polar to equatorial latitudes. Northern species show more cold tolerance than those closer to equatorial regions. It is also generally known that overwintering birds have to confront not only severe cold but also scarcity of food. Long nights reduce foraging to a couple of hours.

The enhanced resistance to cold in birds is achieved primarily by their efficient insulation, behavioural strategies and increased shivering activity (for rev. see Hissa, 1988 and Table 1). During cold nights game birds like black

grouses (*Tetrao tetrix*) and capercaillies (*Tetrao urogallus*) roost in cavities in the snow. In recent years it has been published more and more information about the roosting abilities also among smaller passerines such as tits (*Parus* sp.), redpolls (*Carduelis flammea*), bullfinches (*Pyrrhula pyrrhula*), snow buntings (*Plectrophaenax nivalis*) and yellow buntings (*Emberiza citrinella*). During cold nights small birds like tree creepers (*Acanthis* sp.) and kinglets (*Troglodytes* sp.) huddle together (Löhr, 1955, MacKenzie 1959) and they can also use dense foliage or cavities in trees or other structures.

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Table 1. Response to cold in birds

A. Exothermic (external)

- Changing peripheral blood supply (vasomotor tone)
- Counter-current vascular system (e.g. leg temperature)
- Microhabitat selection
- Postural adjustments
- Ptilomotor (changing the insulative capacity of plumage)
- Huddling
- Roosting in snow

B. Endothermic (internal)

- Locomotor activity
- Shivering
- Non-shivering thermogenesis (NST, regulated by glucagon + some other unknown hormones): Birds do not have brown adipose tissue and consequently sympathetic nervous system does not stimulate heat production in birds as in cold-adapted mammals)

Small passerines may also tolerate cold nights by becoming into hypothermic (Reinertsen, 1983, 1984).

THE MECHANISMS FOR HEAT PRODUCTION

The available data on the mechanisms of thermoregulation and thermogenesis suggest that they differ in many aspects between birds and mammals. Since there are several features which distinguish birds from mammals; e.g. their relative higher energy consumption, higher body temperature (40-42 °C vs. 37-39 °C in mammals), flight with feathered wings and lack of brown adipose tissue (see below); the problem of physiological control of avian body temperature is undoubtedly of considerable physiological interest. We are asking whether birds have the ability to generate heat by non-shivering thermogenesis (NST) as do mammals? This has long been a matter of speculation. Shivering thermogenesis seems to be the only or the main manner for extra heat production in birds. Shivering is an increase in the rate of heat production due to contractile activity of the skeletal muscles (primarily *M. pectoralis major*) not involving voluntary movements or external work (Bligh & Johnson, 1973) but is under voluntary control (Hemingway 1963). In the avian body the pectoralis muscle

constitutes the largest, the most powerful, and metabolically, the most active skeletal muscle. Its weight may constitute up to 20-30 % of the total body weight (Hartman, 1961). Heat production in shivering is produced by splitting ATP to ADP + P_i. During the mechanical work, part of the energy released in this process is converted to external work (e.g. flight); however during intensive shivering this is converted to heat.

Whether shivering occurs in bursts or is continuous seems to vary with the species (see for more details Stevens *et al.*, 1986, Hohtola & Stevens, 1986). In most avian species so far studied shivering is continuous but there are also species which have variable intensity or bursts of shivering. The latter is observed e.g. in the house wren (*Troglodytes aedon*, Odum, 1942), black-capped chickadee (*Parus atricapillus*, Chaplin, 1976), willow ptarmigan (*Lagopus lagopus*, Aulie, 1976) and Japanese quail (*Coturnix coturnix japonica*, Stevens *et al.*, 1986).

NST IN BIRDS

In mammals an increase in sympathetically mediated NST occurs in the course of cold-adaptation. In newly born and cold-acclimated mammals the site of NST and enhanced calorogenic response to noradrenaline (transmitter of sympathetic nervous system) have been con-

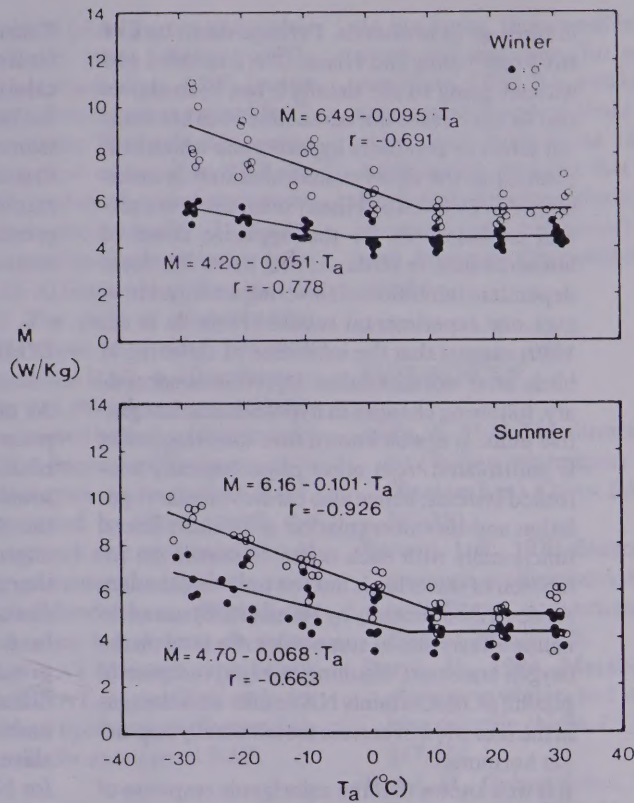


Figure 1. An example of avian (in this case capercaillie, *Tetrao Urogallus*) relation of metabolic rate ($\dot{M}$) to ambient temperature (T_a) in winter (W) and summer (S). The capercaillie is the largest Tetraonidae in Euro-Asia. The sexual dimorphism is also well-known. In this example body weight of females was 1.80 kg in S and 1.85 kg in W and of males 4.00 kg and 4.11 kg, respectively. The lower critical temperature (= the lower limit of thermoneutral zone) was for females +12 °C (S) and +9 °C (W) and for males +6 °C and -3 °C respectively (Rintamäki *et al.*, 1984).

sidered to be primarily brown adipose tissue (BAT) and skeletal muscle. BAT may constitute as much as 60% of NST (Foster & Frydman, 1978, 1979).

Birds living in arctic and subarctic regions are known to have a slightly higher resting metabolic rate than related species or subspecies from warmer climates (for rev. see Hissa, 1988) and this has been correlated to a lower critical temperature in winter (primarily due to improved insulation) (Fig. 1).

Winter-acclimatized birds withstand also cold much longer than summer-acclimatized birds and even smaller passerines can maintain normothermia from 30 minutes to several hours even at -50/-70 °C in winter, whereas in summer

they become hypothermic immediately at these temperatures (Dawson *et al.*, 1983a, b). The winter stamina might be a result of improved capacity for substrate mobilization and delivery to skeletal muscles (= improved shivering). However the main question: do birds have the ability to generate heat as do mammals by stimulating NST has long been a matter of speculation. Do birds have BAT? Does noradrenaline (NA) stimulate heat production in birds as in cold-adapted mammals? Unfortunately very little has been done to elucidate probable mechanisms of regulated NST in birds. However there are plenty of evidence showing that stimulation of sympathetic nervous system or injections of noradrenaline do not improve heat production

in birds as in mammals. Perhaps do to lack of BAT (see below and Hissa, 1988). In short and without going to the details it has been shown that in most cases noradrenaline is either without effect or produces hypothermia which is in contrast to the observations obtained in mammals. As reviewed by Hissa (1988) there are several explanations for the opposite effect of noradrenaline in birds. At first there is a dose-dependent inhibition of shivering activity. However our experimental results (Hohtola *et al.*, 1980) suggest that the inhibition of shivering in birds after noradrenaline injection is secondary, following changes in hypothalamic integrative units. It is well known that shivering reflex is not isolated from other physiologically controlled systems, hence also cardiovascular regulation and thermoregulation are closely linked functionally with each other. However the inhibition of shivering is not the only explanation. As described recently by Hissa (1988) noradrenaline affects also at some unknown level on the oxygen transport resulting in rapid increase of plasma pCO₂. Certainly NA results also changes in the secretion of certain metabolically important hormones.

It is well known that the calorogenic response of mammals to NA are elicited via β -adrenoceptors. Consequently blocking of β -receptors with propranolol affects thermoregulation producing hypothermia. In many papers it has been shown that propranolol affects birds by producing hypothermia. However as shown and discussed recently by Hissa (1988) the hypothermic response of birds to propranolol does not give any evidence of regulated NST in a similar manner as in mammals. The experiments performed with β -receptor blockers show that propranolol affects as follows:

- it blocks directly shivering
- it reduces heart rate and blood pressure
- it stimulates the output of catecholamines (primarily noradrenaline, which produces hypothermia as previously described)
- it affects gas transports
- it increases plasma viscosity (= increased hematocrit values)
- it affects at metabolically important hormone levels.

Taken together it seems obvious that birds differ from mammals in that noradrenaline has no calorogenic effects in birds. Although β -receptor blocker, propranolol, inhibits heat production as in cold-acclimated mammals the dramatic physiological effects of propranolol resulting in hypothermia give no evidence of the presence of noradrenaline (β -receptor) transmitted NST in birds.

DO BIRDS HAVE BAT?

As mentioned above it seems obvious that the presence of NST cannot be totally ruled out in birds. We have shown in our experiments seasonal changes in the resting metabolic rate of the domestic pig, *Columba livia*; higher oxygen consumption in winter than in summer at thermoneutral zone. However, no seasonal changes in EMG-activity was measured (Saarela *et al.*, 1980). Winter-acclimatized blackgrouses (*Lyrurus tetrix*) demonstrated a difference of 10-11 °C between lower critical ambient temperature and the threshold of shivering thermogenesis, and show a capacity for NST 45% above the resting metabolic rate (Rintamäki *et al.*, 1983). Our recent observations (Hohtola *et al.*, unpublished) show that winter-acclimatized pheasants (*Phasianus colchicus*) increase their metabolic rate by 2.8 (males) and 2.0 (females) without any evidence of shivering. Barré (1984) demonstrated a clear metabolic increase 18-27% above the resting metabolic rate of king penguin chicks without shivering as an immediate response to cold (for further examples see for rev. Hissa, 1988).

So, if we assume the existence of NST in birds, how then is it regulated? As mentioned mammals use primarily BAT, dependent upon the action of NA. Generally birds tend to store more fat in winter than in summer and during the winter night the energy reserve is fat (see e.g. Evans, 1969; Blem, 1973). Also the quality of fatty acids show seasonal changes. In winter linoleic acid is predominant but a greater proportion of other unsaturated fatty acids is also typical (e.g. Barnett, 1970; Carey *et al.*, 1978). The ultrastructural studies reveal that the cells

of avian adipose tissue (AAT) are multilocular (Luckenbill & Cogen, 1966; Oliphant, 1983; Barré *et al.*, 1986; Saarela & Hissa, 1988). Its appearance is intermediate between typical BAT and white adipose tissue. The main differences in comparison to typical mammalian BAT are as follows: 1. The number of mitochondria are fewer and also their size is smaller. 2. The cytochrome oxidase activity of mitochondria fraction of AAT is 10-20 times lower than that of isolated BAT. 3. The yield of mitochondrial protein of AAT is about 10-30 times less than in a typical mammalian BAT. 4. Histofluorescence studies do not reveal any sympathetic nerve endings associated with the adipose cells as in typical BAT. A dense capillary network is surrounding adipocytes as in typical BAT. However as concluded by Hissa (1988) the dense capillary network and multilocularity of AAT may merely guarantee the rapid release of fatty acids into the bloodstream, although the stimulatory mechanism is unknown. It seems today obvious that AAT although superficially resembling typical BAT, is modified only for rapid mobilization of lipids but not for production of extra heat *in situ* as a typical BAT.

USE OF ENERGY AT TISSUE LEVEL

The avian muscle cells themselves have limited amounts of endogenous fatty acids and glycogen regardless of the season as shown in cardueline finches (*Carduelis tristis*) by Carey *et al.*, 1978, Marsh & Dawson, 1982. In any cases they probably would be useful in severe shivering only for a few minutes as suggested by Dawson *et al.* (1983a). Consequently summer birds become hypothermic faster than winter birds probably due to their inability to deliver substrate (primarily fatty acids) for skeletal muscles and/or in failing to use fat reserves at cellular level. A clear correlation between shivering thermogenesis and the capacity to mobilize fat is thus obvious. Additionally, there might also be differences at mitochondrial activities as show our recent observations (Saarela & Hissa, 1988).

But what are the stimulatory mechanism? We

do not know. Best candidates are growth hormone and glucagon (for more details see for rev. Hissa, 1988). It is well known that avian glucagon level is several times higher both in pancreas and in plasma than in mammals. Additionally, we know that it has also powerful lipolytic and glycogenolytic properties as shown in experiments performed both *in vivo* and *in vitro*. However this possibility has to be investigated.

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